THESIS FOR M.D. DEGREE.

ANEURYSM OF THE HEPATIC ARTERY.

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ANEURYSM OF THE HEPATIC ARTERY.

By WILLIAM ROLLAND, M.B., Ch.B.,

The extreme rarity of this condition amply justifies the publication of the present case, but apart from this, certain points of interest are presented, especially in the etiology, which may add to our knowledge of this disease. That the condition is an uncommon one is shown by the fact that, after a careful search of the literature, only forty undoubted cases have been collected; this number excludes aneurysms of the pancreatic artery and of the coronary artery of the stomach, which are included by some writers among aneurysms of the hepatic artery, but includes aneurysms of the cystic artery, a branch more closely connected with the liver than those already mentioned. Much speculation has been indulged in regarding the etiology of the condition, and many widely different factors have been suggested. It will be seen that the symptomatology is a very definite one, but that it resembles so closely that of certain other commoner conditions as to render the diagnosis a matter of extreme difficulty.

Extracts from clinical reports.—Mrs. M'L., aged 46, a housewife, was admitted to the Western Infirmary, Glasgow, under the care of the late Sir Thomas M'Call Anderson, on 25th May,

1906, complaining of swelling of the legs and shortness of breath. Her illness commenced four years ago with dyspnœa, which gradually increased in severity. Œdema of the legs was first noticed three months ago. There has been no pain in any part of the body, nor has there been any hæmorrhage.

Examination shows considerable œdema of both legs. There is a large cicatrix with coppery pigmentation on the outer aspect of the left knee; the patient states, on being questioned, that this is the result of an eruption which has been present off and on for the last seven years. The urine is passed in normal amount. It contains abundant albumen and granular and hyaline tube-casts. The lungs show some hypostatic congestion at both bases. The heart is considerably increased in size, especially towards the left, the transverse measurement of the precordial dulness being 7½ inches.

The *diagnosis* was chronic nephritis, with hypertrophy of the heart. The symptoms gradually disappeared from the time of admission, and the patient made an uneventful recovery, being dismissed much improved on 29th June, 1906.

On 7th January, 1907, the patient was readmitted with a return of her previous symptoms. She states that shortly after leaving hospital her feet began to swell, and all her other symptoms returned.

Examination at this time reveals a condition practically similar to that which existed during the previous residence in hospital. There is no jaundice, but some bile pigment is detected in the urine. There is a considerable amount of free fluid in the abdominal cavity. There is no pain in the liver region.

7th February, 1907.—The patient has been improving daily. There has been no hæmatemesis or melæna. The anasarca is much less, and there is very little dyspnœa. At 6·15 P.M. to-day she complained of a sudden, severe pain in the right hypochondrium. She became extremely pallid, and the pulse was slow and very feeble. The respirations gradually became less frequent, and she died at 6·30 P.M.

The *post-mortem* examination was performed by the writer on 9th February, 1907.

The body is very emaciated. The lower limbs are œdematous. There is marked anæmia. On the outer aspect of the left knee-joint is a large cicatrix of a coppery colour; the cicatrix has more or less circular margins.

Thorax.—The serous cavities are normal.

Heart.—Markedly hypertrophied, both right and left ventricles participating. There is moderate dilatation. The

weight is 1 lb. 8 oz. The aortic and pulmonary valves are competent.

Diameter of aortic valve, ...	1	inch.
Mitral, ...	1·3	,,
Tricuspid,.	1·5	,,

Some atheromatous patches of small size on mitral cusps and in aorta. Coronary orifices clear. No endocarditis. Myocardium evidently healthy. In the left ventricle the endocardium is thickened, and there are some subendocardial hæmorrhages.. The maximum thickness of the wall of the left ventricle is 1 inch, of the right ventricle 0·5 inch. There are large outstanding columnæ carneæ in the right ventricle. There is marked thickening and atheroma in the thoracic vessels of large and medium size.

Lungs.—Chronic bronchitis and emphysema. There is an old hæmorrhagic infarction, about half an inch in diameter, in the outer part of the left lower lobe; this is softened in part, and the softened material is seen microscopically to consist of pus containing pneumococci.

Abdomen.—The peritoneal sac contains 3 pints of bloody serum and clotted blood.

Liver.—Weight, 5 lb. It is of pale yellow colour, with red mottling in the centre of the lobules, and is evidently fatty. On the surface it is slightly irregular and granular in parts, but on section it does not appear cirrhotic. Numerous little elevated points, about the size of a pin point, are seen all over Glisson's capsule, and often aggregated into clusters. No gall-stones are found in the gall-bladder. In the centre of the under surface of the left lobe of the liver there is a deep ragged hole, from which the hæmorrhage has proceeded. Around this the capsule of Glisson has been stripped and raised into a blister, about 5 inches in diameter, occupying the greater part of the under surface of the lobe. An antero-posterior incision was made through the liver, traversing the rupture obliquely. Recent blood-clot was picked out of the rupture and the vessels examined, and finally, in the depths of the part of the rupture which remained in the left half of the liver, a true aneurysm of fusiform shape, about 2·5 cm. in length, by 1·25 cm. in diameter, was found. The vessel on which it was situated was traced back to the portal fissure, and identified as hepatic artery. A rupture, about 3 mm. long, was apparent in one side of the sac. The aneurysm is situated within the parenchyma of the left lobe of the liver (Fig. 1). The vessel from which it sprang is the

main branch to the left lobe of the liver. The aneurysm is situated 10 or 11 cm. from the bifurcation of the hepatic artery. The vessel at the point of rupture is barely 2 mm. in diameter. A small aneurysm, about the size of a pea, was found in the portal fissure close to the main trunk of the portal vein and hepatic artery; this was quite filled with dense clot. Another, about the size of a barleycorn, was found close to the main trunk from which the ruptured aneurysm sprang; this was also near the portal fissure, and was completely occluded by thrombus. Both of these small aneurysms were also intrahepatic. No other aneurysm was found in the abdominal vessels. The amount of atheroma in the abdominal aorta and large vessels of the abdomen is small.

Spleen.—Weight, 6 oz.; somewhat enlarged; soft; dark coloured.

Kidneys.—Well-marked subacute nephritis. Thick, irregular cortex. Slightly granular on surface. Vessels thickened.

Suprarenals.—Large and very yellow, as if fatty.

The *histological appearances* of the kidneys, lungs, and liver were investigated, when the following observations were made:—

Kidneys.—Cortex increased in thickness. Surface depressed irregularly at places, by fibrous bands. Increase of rather cellular fibrous tissue of patchy distribution, and accompanied by infiltration of lymphocytes and large hyaline cells. In some parts many glomeruli are aggregated together. Most of the glomeruli are of normal appearance, but many show varying degrees of fibrosis, affecting both the tuft and the capsule of Bowman. The tubular epithelium shows degrees of degeneration, varying from cloudy swelling to complete necrosis. The amount of catarrh is considerable, and in many parts evidences of compensatory proliferation can be seen. The renal artery shows some thickening of the media, with relatively increased fibrous tissue, but the tunica intima appears normal. The collecting tubules contain many hyaline casts. The appearances are those of a subacute nephritis, with early interstitial changes.

Lung (sections of infarction).—The sections show an infarction of some considerable duration; the pleura over it is thickened, and surrounding it is a layer of dense fibrous tissue; organisation changes are far advanced, and many large cells, full of hæmosiderin granules, are seen. The adjacent lung tissue shows much catarrh and some complementary emphysema. One part of the infarction is the

seat of a suppurative softening; in this part pneumococci can be seen in the sections stained by Gram's method.

Liver.—Throughout the organ a subacute interstitial hepatitis is present; the interlobular tissue varies greatly in cellularity, some parts consisting almost entirely of an infiltration of lymphocytes and epithelioid-like cells, while others are much more fibrous (Fig. 2). The little elevated points on Glisson's capsule, noted at the autopsy, are seen to be tags of dense fibrous tissue; their significance is not quite apparent. The branches of the hepatic artery, and especially the smaller branches, show very definite changes (Figs. 3, 4, 5, and 6). In those which are typically affected, the wall presents a hyaline, homogeneous appearance, the differentiation into coats being more or less lost. It was proved by the staining reaction that this was not due to amyloid degeneration. The intima shows varying degrees of endarteritis obliterans, there being great cellular proliferation without any degeneration; this in some vessels is so extreme as to cause almost complete obliteration of the lumen. In all the affected vessels a well-marked periarteritis is present. This is shown by a zone of tissue, resembling granulation tissue, completely surrounding the vessel wall; the cellular elements present are chiefly large epithelioid cells and polymorphonuclear leucocytes, with a few mononuclear leucocytes. This periarterial infiltration in some cases fades gradually into the surrounding liver tissue, while in others there is a zone of young fibrous tissue around it. In many vessels this granulation tissue appears to be invading the degenerating vessel wall. There are thrombi in some of the affected vessels. The larger vessels do not show these changes, but most of them present an irregular thickening of the intima. Numerous pieces of liver tissue were treated by the silver impregnation method, and examined for spirochæta pallida, but in none of them was a positive result obtained. Sections of the medium-sized aneurysm showed in its wall portions of the original vessel wall more or less necrosed, and having a layer of granulation tissue outside (Fig. 7). Serial sections of the smallest aneurysm showed it to be a fusiform aneurysm, and, at the same time, the vessel on which it was situated was seen to present changes similar to those just described (Fig. 4). In drawing up the subjoined list of cases, the tabular method employed by De Vecchi has been adopted, as it shows at a glance what are the outstanding features of each case.

No.	Date.	Author.	Publication.	Age.	Sex.	Number	Size.	Position.	Immediate Cause of Death.	Etiology.	Symptoms.	Post-mortem Appearances.
1	1809	Wilson.	Lectures on Blood, etc., before R. C. of Surgeons, 1819, p. 379. (Specimen now in Surg. Hall, Edinburgh.)	50	M.	One.	Resembles heart in size and shape.	Left branch; intrahepatic.	Rupture.	?	?	?
2	1821	Pitcairn.	Specimen in Museum of Surg. Hall, Edinburgh, 36-296 (evidently unpublished).	48	M.	One.	Hen's egg.	Main trunk; extrahepatic.	Rupture into peritoneal cavity.	?	None.	?
3	1833	Sestier.	Bulletins de la Société Anatomique, tom. viii, p. 30.	?	F.	One.	Hazelnut.	Right branch; extrahepatic.	Exhaustion.	?	"Chronic painful affection of liver."	Gangrene of gall-bladder.
4	1834	Stokes.	Dublin Med. Journ., 1st series, vol. v, p. 401.	35	M.	One.	Large orange.	Main trunk; extrahepatic.	Rupture into peritoneal cavity.	?	Jaundice; hæmatemesis; slight pain.	?
5	1834	Jackson.	Med. Mag., Boston, vol. iii, p. 115.	22	M.	One.	Pullet's egg.	Main trunk; extrahepatic.	Rupture into hepatic duct.	?	Pain; jaundice.	Phthisis pulmonalis; old carotid aneurysm.
6	1855	Lebert.	Anatomie Pathologique, tom. ii, p. 322.	30	F.	One.	Pigeon's egg.	Main trunk; extrahepatic.	Rupture into gall-bladder.	Embolic?	Pain; slight jaundice; hæmatemesis; melæna; anæmia.	Old mitral endocarditis; old and recent pericarditis.
7	..	Lebert.	Quoted by Uhlig, Krankheiten der Arterien.	?	M.	One.	Hazelnut.	Right branch.	?	?	"Gastric disturbance."	?
8	1856	Ledieu.	Journ. de. Med. de Bordeaux, Mars, 1856.	54	F.	One.	Hazelnut.	Main trunk, just above origin of pyloric artery.	Albuminuria and general dropsy.	?	None caused by aneurysm.	Cavity quite occluded by very firm coagulum.
9	1858	Wallmann.	Virchow's Arch., Bd. xiv.	36	F.	One.	Child's head.	Main trunk, 7 mm. above subdivision.	Rupture into peritoneal cavity.	?	Very severe paroxysmal pain; jaundice; no fever.	Gall-bladder much distended.
10	1868	Uhlig.	Inaug. Diss., Leipzig, 1868.	48	M.	One.	Goose's egg.	Left branch; extrahepatic.	Rupture into peritoneal cavity.	Infective? (osteomyelitis). Atheroma?	Severe pain.	?
11	1871	..	Specimen in St. George's Hospital Museum, VI, 86A (mentioned by Rolleston, Diseases of Liver, etc., 1904, p. 44).	35	M.	One.	Cricket ball.	Main trunk; extrahepatic.	Rupture into peritoneal cavity.	Atheroma in aorta?	Dead on admission to hospital.	Adherent to small intestine.
12	1871	Quincke.	Berlin. klin. Wochen., 1871, p. 349.	25	M.	One.	Chestnut.	Right branch; intrahepatic.	Exhaustion from loss of blood.	Infective?	Severe pain; hæmatemesis; melæna; jaundice; fever.	Pneumonia; no rupture found.
13	1875	Standhartner.	Ber. des. Wiener allg. Krankenh., 1875.	23	M.	Two.	(1) Walnut. (2) Small nut.	(1) Right branch (2) Left branch.	Rupture of larger aneurysm into peritoneal cavity.	Infective?	Icterus; high fever (not due to aneurysm).	Suppurative mediastinitis; pleurisy; pneumothorax.
14	1877	Ross and Osler.	Canadian Med. Journ., vol. vi, July, 1877.	21	M.	One.	Small lemon.	Right branch; extrahepatic.	Pyæmia.	Infective?	Those of pyæmia.	Double pleurisy; abscesses of liver.
15	1878	Borchers.	Inaug. Diss., Kiel, 1878.	17	M.	Two.	(1) 2·2 cm. in diameter. (2) 2·5 cm.	Intrahepatic.	"Amputation of femur."	Infective? (osteomyelitis). Trauma?	Pain; icterus; hæmatemesis; melæna; fever.	Ruptured into hepatic duct.
16	1878	Irvine.	Trans. Path. Soc., London, 1878, vol. xxix, p. 128.	45	M.	One.	Small almond.	Left branch; intrahepatic.	Hæmorrhage into stomach.	Infective.	Slight pain; hæmatemesis; melæna.	Stomach adhering to, and communicating with, abscess of liver; early cirrhosis of liver.
17	1880	Drasche.	Wiener med. Wochen., 1880, No. 37-39.	27	M.	One.	Hazelnut.	Right branch, 1·5 cm. beyond division.	Rupture into peritoneal cavity.	?	Severe pain; hæmatemesis.	
18	?	Heschl.	Quoted by Drasche. Specimen in Gratz Museum.	56	F.	One.	Pigeon's egg.	Main trunk, just before division.	Tuberculosis.	?	None.	
19	1883	Weinlechner.	Aerztl. Bericht. der K. K. Krankenh. zu Wien, S. 269.	Young	M.	One.	?	Main trunk.	Rupture into peritoneal cavity.	Infective?		Recent osteomyelitis of femur.
20	1885	Chiari.	Prag. med. Wochen., 1885, No. 4.	33	M.	Three.	Largest, 2×1 cm.	Cystic artery.	Rupture into gall-bladder.	Cholelithiasis.	Hæmatemesis; melæna.	Fistula between gall-bladder and duodenum.

No.	Date.	Author.	Publication.	Age.	Sex.	Number	Size.	Position.	Immediate Cause of Death.	Etiology.	Symptoms.	Post-mortem Appearances.
21	1886	Caton.	Clin. Soc. Trans., 1886, vol. xix, p. 275.	40	M.	One.	1 inch in diameter.	Main trunk; extrahepatic.	Rupture into common duct.	?	Intense pain; jaundice; hæmatemesis; melæna; fever.	Other organs healthy.
22	1892	Hale White.	Brit. Med. Journ., Jan., 1892, p. 223.	18	M.	Two.	(1) Tangerine orange. (2) Slightly smaller.	(1) Right branch extrahepatic. (2) Left branch; intrahepatic.	Rupture of (1) into peritoneal cavity.	Infective?	Pain; jaundice; hectic temperature.	Pneumonia; empyema.
23	1892	Sachs.	Deutsch. med. Wochen., No. 20, S. 443.	60	M.	One.	?	?	Rupture into portal vein. Aneurysmal varix.	?	?	Numerous gastric ulcers; portal thrombosis.
24	1893	Sauerteig.	Inaug. Diss., Jena, 1893.	31	M.	Two.	(1) Apple. (2) Cherry.	1) Right branch extrahepatic. (2) Left branch; intrahepatic.	Rupture of (1) into cystic duct.	?	Severe pain; jaundice; fever; hæmatemesis; melæna.	Operated on for cholelithiasis.
25	1893	Ahrens.	Inaug. Diss., Greifswald, 1893.	32	F.	One.	Hen's egg.	Main trunk.	Rupture into peritoneal cavity.	Infective.	Collapse.	Phlegmonous inflammation of leg; hæmatoma of hepato-duodenal ligament.
26	1894	Schmidt.	Deutsch. Archiv. f. Klin. Med., 1894, Bd. lii, p. 536.	40	F.	One.	?	Right branch; extrahepatic.	Rupture into hepatic duct.	Cholelithiasis.	Pain; hæmatemesis; melæna; jaundice.	Perforations between gall-bladder and duodenum.
27	1894	Niewerth.	Inaug. Diss., Kiel, 1894.	19	M.	Two.	(1) 1·8×3 cm. (2) Cherry.	1) At the point of division. (2) Main trunk.	Rupture of (1) into gall-bladder and peritoneal cavity.	Atheroma of hepatic artery.	?	Cirrhosis of kidney; atheroma of aorta, etc.
28	1895	Mester.	Zeitschr. f. Klin. Med., 1895, Bd. xxviii, p. 93.	42	M.	One.	5×3·5 cm.	Right branch; intrahepatic.	Rupture into hepatic duct.	Trauma.	Pain; fever; hæmatemesis; melæna; jaundice.	Fatty degeneration of heart; broncho-pneumonia; anæmia.
29	1896	Sainton (Bernard's Thesis, 1897).	Société Anatomique, Paris, May, 1896.	46	M.	One.	Large orange.	Main trunk.	Rupture into peritoneal cavity.	Diffuse Atheroma.	Slight jaundice; collapse.	Atheroma of aorta, etc.
30	1897	Hansson.	Centralb. f. die Grenzgebiete der Med. u. Chir., i, p. 299.	14	M.	One.	Hen's egg.	Right branch; intrahepatic.	Rupture into hepatic duct.	Infective? (osteomyelitis).	Hæmatemesis; melæna.	?
31	1900	Sacquépée.	Zentralb. f. Path. Anat., 1900, Bd. xi, S. 748.	44	M.	One.	Orange.	Intrahepatic.	Rupture into peritoneal cavity.	Syphilitic endarteritis.	?	Cirrhosis of liver.
32	1901	Brion (quoted by Grunert).	Deutsch. Aerzte Zeitung, No. 18.	..	..	..	..	..	..	..	..	..
33	1902	Sommer.	Prag. med. Wochen., Bd. xxvii, H. 38, S. 469.	28	M.	One.	?	Main trunk.	Perforation into common duct.	Infective.	Icterus.	Pneumonia; no atheroma.
34	1902	Sommer.	Prag. med. Wochen., Bd. xxvii, H. 38, S. 469.	65	F.	One.	?	Point of origin of gastro-duodenal artery.	Rupture into duodenum and peritoneum.	Trauma.	No icterus.	No atheroma.
35	1903	Kehr.	Münch. med. Wochen., 1903, p. 1861.	29	M.	One.	Hen's egg.	Right branch; extrahepatic.	(Recovery after operation.)	?	Pain; jaundice; hæmatemesis.	?
36	1904	Grunert.	Deutsch. Zeitschr., f. Chir., Bd. lxxi, S. 158.	21	M.	One.	App'e.	Main trunk.	?	Infective? Gall-stones?	Those of gall-stones.	Operation; gall-bladder full of calculi.
37	1905	De Vecchi.	Bull. della Scienze med. di Bologna, 1905.	83	M.	One.	Cherry.	Right branch; extrahepatic.	Cerebral hæmorrhage.	Gall-stones?	None caused by aneurysm.	Jaundice: cerebral hæmorrhage; granular kidney; gall-stone.
38	1906	Alessandri.	Bull. d. v. Accad. med. di Roma; Bd. xxxii, p. 63.	22	M.	One.	Hen's egg.	Right branch; extrahepatic.	Rupture into bile ducts.	Infective? (pneumonia).	No pain; no fever; jaundice; melæna; liver enlarged; gall-bladder palpable.	?
39	1906	Livierato.	Gazz. d. Osp. Milano, 1906, Bd. xxvii, p. 593.	28	M.	One.	..	Main trunk.	Rupture into peritoneal cavity.	Infective? (pneumonia).	Severe pain; vomiting; collapse.	Double pneumonia: thrombosis of hepatic artery; liver healthy.
40	1906	Wätzold.	Münch. med. Wochen., liii, p. 2107.	44	M.	Several.	Largest, a cherry; others smaller.	Right branch; intrahepatic.	Rupture into peritoneal cavity.	Syphilitic endarteritis and periarteritis.	Pain in liver region; those of cirrhosis of liver.	Cirrhosis of liver; parenchymatous nephritis; endocarditis of aortic valve.

Pathological anatomy.—The most generally accepted and most natural subdivision of the cases is into extrahepatic and intrahepatic; the majority of the cases belong to the former division. Of the 40 cases which have been collected, 24 were extrahepatic and 8 were intrahepatic, while in 2 cases, in each of which 2 aneurysms existed, one of the aneurysms was extrahepatic while the other was intrahepatic. In 6 of the cases, the relation of the aneurysm to the hepatic parenchyma is not stated. In the present case there were three aneurysms, all of which were intrahepatic. The main trunk of the artery has been affected more often than either of its branches; in 16 of the cases the main vessel was involved; in 12 the aneurysm was on the right branch, and in 3 on the left; in 3 cases there was an aneurysm on each of the branches. In 1 case the aneurysm was on the cystic artery. In the author's case, the main aneurysm was on the left branch. Schmidt and Mester both mention that in their cases the aneurysm was of the false variety, but the other authors do not state to which class the aneurysm belonged. In the present instance all the three aneurysms were of the "true" variety. Of the 40 cases, 32 resulted in rupture of the sac, 6 were found unruptured, while in 2 of the cases the condition of the aneurysm is not stated. The rupture occurs in the great majority of cases into either the peritoneal cavity or the bile passages. In 16 cases rupture took place into the peritoneal cavity, in 13 into the bile passages, and in 3 into other organs, viz., the stomach, duodenum, and portal vein. In the author's case the main aneurysm ruptured into the peritonéal cavity. That the aneurysm may undergo spontaneous healing is shown by Ledieu's case, in which an aneurysm occluded by thrombus was found on the hepatic artery of a patient, who died of renal disease without any symptoms referable to the liver. In the case reported by Ross and Osler, the patient died with symptoms of pyæmia, and the liver was found on section to contain multiple abscesses. The aneurysms rarely attain a large size. Those which are extrahepatic are generally larger than the intrahepatic. The largest one hitherto recorded is that of Wallmann, which was of the size of a child's head, while the smallest of the three in the present case—viz., that which was the size of a barleycorn—is probably the smallest which has been put on record. The average size appears to be about equal to that of a hen's egg. In 32 of the 40 cases only one aneurysm was present; in one case the number could not be determined; in five cases (those of Standhartner, Borchers,

Hale White, Sauerteig, and Niewerth) two aneurysms were found; in the case of Chiari there were three aneurysms present; in the author's case there were also three, while in Wätzold's there were several aneurysms. It is of interest to note that in all cases of intrahepatic aneurysm rupturing into the peritoneal cavity, there is of necessity a rupture of the liver produced. In a cursory examination this appears like an ordinary traumatic rupture, except that Glisson's capsule is apt to be stripped from the liver, and careful dissection is required to determine the true cause of the lesion. One can imagine what far reaching effects a case of this kind might have, if occurring in asylum practice, where there was any question of violence having been used in restraining the patient. Sacquépée was the first to point out the importance of aneurysm of the hepatic artery as a cause of rupture of the liver, and Wätzold has subsequently published a similar case. The only other case of this kind in the literature is the first case of aneurysm of the hepatic artery ever published—viz., that of Wilson—in which the rupture was on the surface of the left lobe. The present publication brings the number of cases of rupture of the liver caused by aneurysm up to four.

Symptoms.—The three symptoms which are most constant in their occurrence are pain, icterus, and hæmorrhage; the hæmorrhage occurs either from the stomach or the intestine.

Pain is the symptom which is more frequently present than any other. Some writers go so far as to say that it is never absent, but it is certain that in some cases only slight pain is present (Stokes, Irvine), while others may reach a fatal termination without this or any other symptom having asserted itself. In the case now recorded, no symptoms of the condition were present until a few minutes before death, when severe pain was felt in the right hypochondrium; this, however, was probably due to the escape of blood into the substance of the liver, and the stripping of the capsule of Glisson, rather than to the mere presence of the aneurysm. In typical cases the pain comes on in paroxysms, and is of great severity, resembling that of biliary colic; Wallman's patient was rendered almost maniacal by it. It is referred to the right hypochondriac and epigastric regions. In the intervals between the paroxysms, pain is usually absent. Many of these paroxysms may occur during the course of the disease. In none of the cases is the incidence of jaundice stated to have had any relation to the attacks of pain. The pain is said to be due to the pressure of the sac on the

FIG. 1.

Photograph of the ruptured aneurysm after dissection. The thin probe lies in the artery, the thick one in the rupture in the aneurysmal sac. The surrounding liver substance is torn. Glisson's capsule is extensively stripped from the surface.

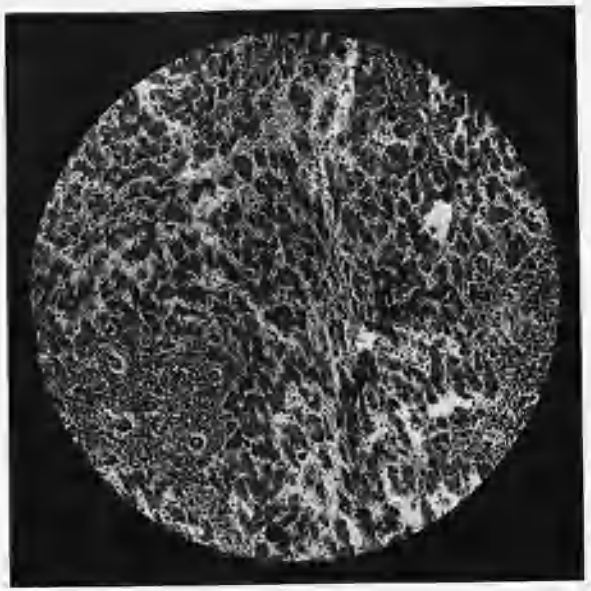

FIG. 2.

Showing the form of interstitial hepatitis which was present throughout the liver. (× 50.)

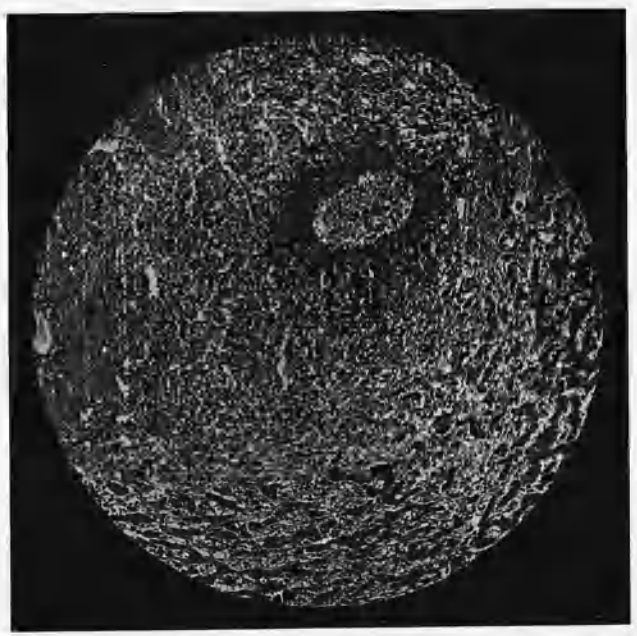

FIG. 3.

A small branch of hepatic artery showing the changes in its wall and in the surrounding tissue. The lumen is occupied by a recent thrombus. Note the extensive periarterial proliferation. (× 50.)

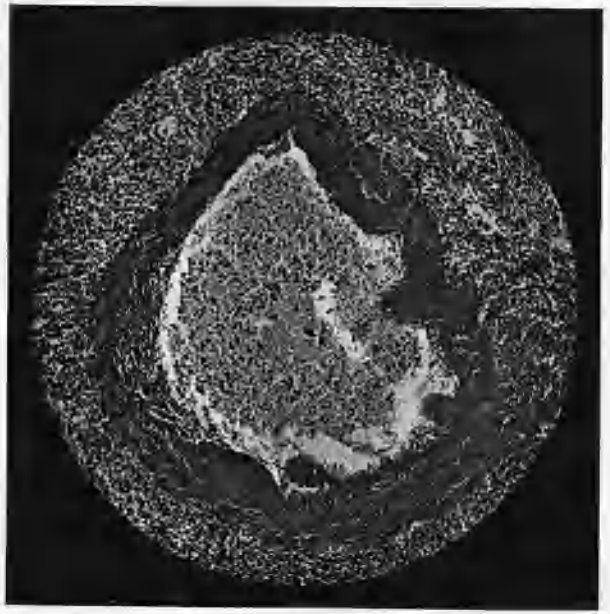

FIG. 4.

Section showing the vessel on which the smallest aneurysm was situated. The periarteritis and degeneration of the vessel wall are well seen. The lumen is occupied by recent thrombus, with, at the lower part, a portion of white thrombus. (× 50.)

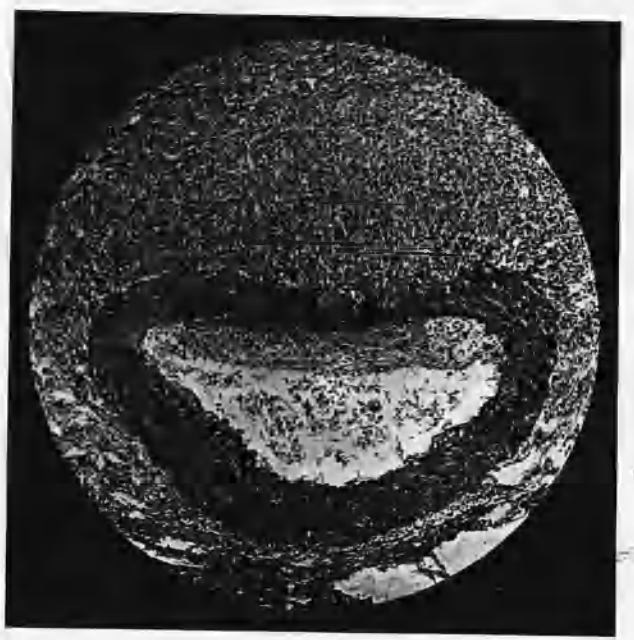

FIG. 5.

A section of hepatic artery showing the disease affecting one side only. The internal elastic lamina stands out as a black line, the section having been stained with Weigert's elastic tissue stain. The wall is much attenuated by invasion with granulation tissue, and the intima at the affected part is much proliferated. (× 50.)

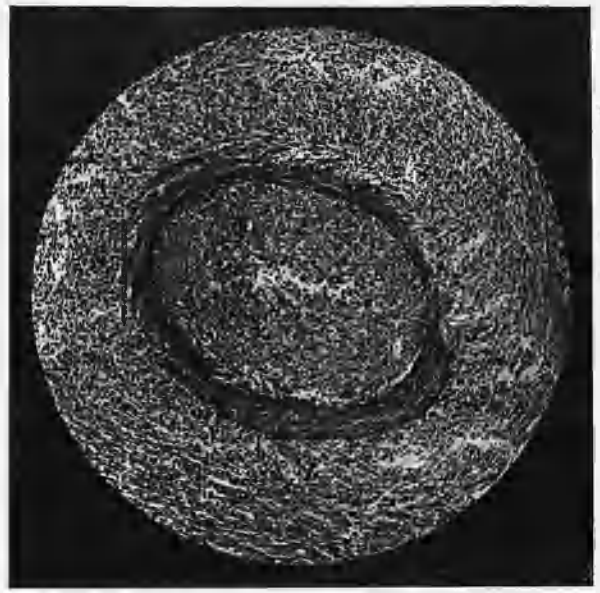

FIG. 6.

A vessel showing changes similar to the others, but in a more extreme degree. The wall is greatly thinned and very degenerate, and the lumen is completely obliterated by proliferation of the cells of the intima. Periarteritis is also well marked. Stained by elastic tissue stain. (× 50.)

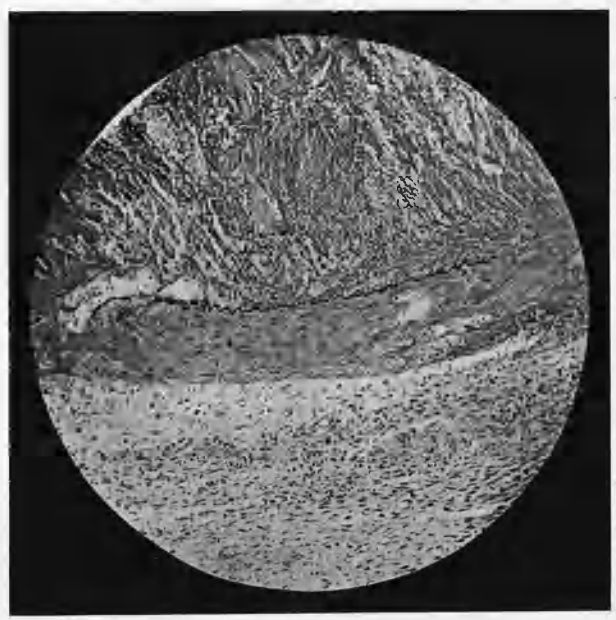

FIG. 7.

A section of part of the wall of the second aneurysm, showing a portion of diseased vessel wall entering into its formation. Above, the dense thrombus which filled the sac is seen, while below there is granulation tissue which surrounded the aneurysm. The internal elastic lamina is plainly seen as a sinuous black line. (× 50.)

branches of the hepatic plexus of nerves. Mester suggests that it is due in intrahepatic cases to pressure on the liver substance or to stretching of Glisson's capsule; the present case appears to support the latter observation. The pain is usually accompanied by tenderness on pressure in the same region as that in which the pain is complained of.

Jaundice occurs in a large proportion of the cases. Of the 40 cases collected, it is definitely stated to have been present in 16, but in most of the others no definite statement is made regarding it. It is significant that it was noted in most of the cases which have been fully reported. In the cases of Quincke, Lebert (1), and Mester, the jaundice was temporary, but in the others more or less permanent. It is in all cases a hepatogenous jaundice, and arises from direct pressure on the hepatic ducts or on the common bile-duct.

Hæmorrhage into the alimentary tract occurs frequently, and gives rise to hæmatemesis or to melæna, or more commonly to both combined. The blood reaches the intestine in a great majority of the cases by way of the bile-duct, and is a direct sequel to a rupture of the aneurysmal sac into the biliary passages. In Irvine's case, however, the rupture occurred into the stomach, and in Sommer's (2) into the duodenum. In rare cases the hæmorrhage may be due to pressure on the portal vein and consequent passive congestion. In 17 of the cases collected, hæmorrhage is noted as one of the symptoms. A striking feature about these hæmorrhages is that they recur frequently with varying intervals between them; death never occurs from a single large hæmorrhage into the bile passages. Mester suggests in explanation of this, that by the rapid filling of the bile passages by blood, the bleeding is itself checked. When repeated hæmorrhages occur a high degree of anæmia is the result.

Fever has been noted in a few cases, and it may be accompanied by rigors. It usually occurs when the paroxysm of pain is at its height. The temperature may be as high as 104° F. This symptom was first noted by Quincke, and, besides being present in his case, it has occurred in those of Standhartner, Borchers, Caton, Sauerteig, and Mester; but in some of these it could be explained by other causes than the aneurysm. Quincke compares these febrile attacks to those which sometimes accompany biliary colic.

The liver may be greatly enlarged in cases where the aneurysm attains a considerable size; this enlargement was present, and was detected during life in the cases of Sauerteig and Wallmann, but it is quite exceptional. Distension of the

gall-bladder has also been noted in one or two cases; in the cases of Stokes and Alessandri, it was due to pressure of the aneurysm on the bile-ducts and retention of bile in the gall-bladder; in Niewerth's case it resulted from hæmorrhage into the gall-bladder. In no case has a pulsating tumour been detected during life, as can easily be understood when one bears in mind the deep position of the extrahepatic aneurysms and the presence of the hepatic parenchyma round the small intrahepatic ones. Rovighi (*Riv. clin. di Bolonga*, 1886, No. 52) diagnosed aneurysm of the hepatic artery in a case in which a systolic murmur was heard over the liver, but the result of the autopsy did not confirm the diagnosis. This symptom has not been observed in any case in which an aneurysm existed. Digestive disturbance is usually present in greater or less degree, and can be explained by the local mechanical effects of the aneurysm, *e.g.*, pressure on the stomach, pressure on the portal vein causing passive congestion, or pressure on the bile-duct causing diminution of the amount of bile entering the intestine. When the aneurysm ruptures into the peritoneal cavity, there are the usual symptoms of internal hæmorrhage with rapidly fatal termination.

Diagnosis.—No case has yet been recognised during life except by means of an exploratory incision. Kehr was the first to diagnose the condition by this means. The two conditions for which it is most likely to be mistaken are cholelithiasis and duodenal ulcer. In cholelithiasis hæmorrhages undoubtedly do occur, though they are relatively much less frequent than in aneurysm. Mester points out that in aneurysm hæmorrhage is often the first symptom observed, whereas in gall-stones it is always a late symptom, and is usually due to fistula formation. The presence of one of the commoner etiological factors of aneurysm would strengthen the diagnosis. One must remember that gall-stones and aneurysm of the hepatic artery frequently exist together, as in the cases of Chiari, Schmidt, Grunert, and De Vecchi. From duodenal ulcer the diagnosis appears to be equally difficult. This is especially so, as duodenal ulcer may cause icterus, either because it happens to be situated on the bile papilla or because of a concurrent catarrh of the common duct. In many cases, therefore, the picture of a duodenal ulcer is reproduced almost perfectly, and the diagnosis would only be possible by exploratory incision.

Etiology.—The condition has occurred in females in only one-fourth of the cases; thus, out of 39 cases in which the

sex was recorded, 30 were in men and only 9 in women. The age of the patients ranged from 14 years (Hansson) to 83 years (De Vecchi). The average age of 36 cases was 37 years. The average age of 8 female cases was 45 years, while the average age of 28 male cases was 35 years. The occupation of the patients is not stated in many of the cases, but those in which it is mentioned suffice to show that the condition is met with under widely different circumstances as regards environment; thus one patient was a gentleman of means, another a clergyman, and another a doctor, while among others we find such occupations as seaman, waiter, pedlar, coachman, tapster, and soldier. Fifteen cases have been published in Germany, 7 in Great Britain and Ireland, 7 in Austria, 6 in France, 3 in Italy, and 1 each in Canada and the United States. Trauma was considered the direct cause of the aneurysm in Mester's case, the patient having been kicked on the abdomen by a horse; the fact that the aneurysm was of the false variety seems to support this view of the etiology. In the case of Borchers also there was a history of injury. The traumatic force may, however, act from the interior of the body, and this is the case in cholelithiasis. In four of the recorded cases gall-stones were present, and in at least two of these (Schmidt and Chiari) their causal relationship seems to have been established. Rolleston (*Diseases of Liver*, p. 44) points out that the proportion of women affected is much higher than is the case in aneurysm of other arteries, and he suggests that, considering the greater frequency of cholelithiasis in females than in males, many of these cases may have depended on gall-stones. It will be seen from my figures that the average age of the males affected (35) corresponds to the decennial period during which aneurysms are most frequently found (30 to 40 years); the average age of the females affected is, however, 10 years greater (45), and since the great majority of the cases of gall-stones occur after the fortieth year, this would appear to lend further support to the view that cholelithiasis may have a direct causal relationship to the condition. The production of an aneurysm under these circumstances is explained by the direct injury of the arterial wall by the gall-stone. This would tend to the formation of a false rather than of a true aneurysm, and it is interesting to note that in Schmidt's case the aneurysm was of the false variety. Another form of injury to the vessel wall from without is illustrated by the case of Irvine, in which an aneurysm formed in the wall of an abscess cavity, after the manner in which aneurysms are formed in phthisical

cavities in the lungs. Embolism, and especially infective embolism, would appear to be a probable cause in many cases. In at least two cases a possible source of simple embolism was found in a valvular endocarditis, while the number of cases preceded by suppurative conditions is very striking. Thus no fewer than four cases were preceded by osteomyelitis, three by pneumonia, one by suppurative mediastinitis, one by pleurisy, one by empyema, and one by phlegmonous inflammation of the leg. It has not yet, however, been proved in any case that the aneurysm was due to metastasis of infective material. Atheromatous changes in the aorta and the larger vessels have been present in a considerable number of the cases examined. Niewerth and Chiari mention that atheroma was present in the hepatic artery itself. These arterial changes are looked upon by most of the authors as of considerable importance; but, as De Vecchi observes, they are never present in the hepatic artery alone, but affect equally most of the other vessels, so that some local condition must be present to determine the production of the aneurysm in the liver. This local condition he believes to be cholelithiasis. Syphilis has been mentioned from time to time as a causal factor, but the publication of Sacquépée's case, and more recently of Wätzold's, has demonstrated clearly how syphilitic infection produces aneurysmal dilatation of the hepatic artery. Unfortunately, I have not had access to the former's original article, but, according to Wätzold, the condition in the arteries was described as a severe endarteritis, which was due to a pre-existing syphilis. In Wätzold's own case the arterial changes are thus described:—"The arteries show a definite thickening of the intima and adventitia; the latter consists mostly of a broad circle of very cellular connective tissue. . . . Almost all the vessels show a considerable thickening of the intima, which in many cases has resulted almost or quite in obliteration." In this case also the changes were almost certainly due to syphilis. In the author's case the alterations in the arteries were practically identical with those described by Wätzold, while there was in addition the hyaline degeneration of the vessel walls. Here, too, the patient was undoubtedly the subject of tertiary syphilis, the patch of ulceration on the left leg being typical of that condition. Between these three cases, all of which occurred in syphilitic subjects, there are other points of resemblance; they were all about the same age; in all, the aneurysm was intrahepatic, and caused rupture of the liver by bursting into the peritoneal cavity; in Wätzold's case, and

in mine, the aneurysms were multiple; in each case there was an early cirrhosis of the liver, due probably to the direct action of the syphilitic virus. These three cases seem to prove conclusively that the branches of the hepatic artery may be the seat of a definite pan-arteritis, and that this is undoubtedly one of the causes of aneurysm of the hepatic artery.

While it can hardly be stated unequivocally that the disease in this case is of syphilitic origin, there can be no doubt that the aneurysmal dilatations are due to visible damage of the arterial wall in association with marked lesions in the surrounding connective tissue. The serial sections of the smallest aneurysm form the most convincing proof of this; the wall of the vessel entering it is completely necrosed, and surrounded by a broad zone of granulation tissue. While the actual necrosis must of itself greatly weaken the resistance of the vessel wall to the blood pressure, this is still further diminished by the invasion of the wall by the surrounding granulation tissue (Fig. 4). The cause of the aneurysm is thus a local condition affecting the branches of the hepatic artery in the liver substance. No similar changes were found in the vessels of any of the other organs examined. De Vecchi states that the production of hepatic aneurysm will always be determined by a local condition, and that usually this condition is cholelithiasis. Here we have a local condition of quite a different nature. It is to be noted that we have not simply a disease of the branches of the hepatic artery, but also a condition which might be called a subacute interstitial hepatitis. The lesions might be explained on the supposition that some virus (syphilitic?) was spreading in the connective tissue, and that this acted on the artery walls from the outside.

Treatment.—Medical treatment is obviously futile, except as a purely palliative means. If the condition is diagnosed during life an operation should be undertaken, with the view of ligaturing the hepatic artery, or one of its main branches, as this appears to be the only procedure which offers the patient any chance of recovery. The case of Ledieu proves that, when the hepatic artery is gradually occluded, a sufficient anastomotic circulation may be developed to supply the liver with arterial blood. Experiments on animals performed by Cohnheim and Litten further proved that the liver in dogs may survive after ligature of the hepatic artery. The artery was first ligatured in man by Kehr, and the result has established the operation as a justifiable surgical procedure in

these cases. Previous to Kehr's case, three others had been submitted to operation (Sauerteig, Niewerth, Mester), but in none of these was the artery ligatured, nor in any case did the patient recover. Thus Kehr's case, besides being the first in which the hepatic artery was ligatured, was the first which was correctly diagnosed during life, and the first in which the patient recovered. Since then other two cases have received operative treatment, viz., those of Grunert and Alessandri; the former case was diagnosed as cholelithiasis, and the latter as biliary obstruction, probably due to tumour of the head of the pancreas. Unfortunately, in neither of these cases was the fatal issue avoided.